Head Injuries in Infants and Young Children

ROBERT H. PUDENZ, *M.D.*, EDWIN M. TODD, *M.D.*, and C. HUNTER SHELDEN, *M.D.*, Pasadena

• **Head injury in infants and young children may produce lesions that are relatively unique for this age group. The uniqueness is generally due to the structural immaturity of the skull, meninges and brain.**

"Derby-hat" and diastatic fractures are common in this age group. Spurious meningoceles result from tearing of the dura which is closely adherent to the skull. The syndrome of "delayed" concussion is more commonly manifested in children. Extradural and subdural hemorrhage may develop from lacerations of the major venous sinuses. A classical extradural hematoma may occur in the absence of fracture across meningeal arterial channels.

The management of patients with head injury has been improved by the more frequent use of tracheotomy, hypothermic techniques and drugs of the "lytic cocktail." Solutions of urea in 10 per cent invert sugar are administered intravenously to control cerebral edema in selected patients.

Head injuries in infants and children of pre-school age have many unique features that distinguish them from those occurring in older children and adults. These differences are due largely to the structural qualities of the skull and the immaturity of the central nervous system in this age group. This report will deal with those facets of the head injury problem that are of greatest importance to the pediatrician. Particular stress will be placed on the management of "closed" injuries not requiring surgical treatment.

ANATOMICAL AND PATHOLOGICAL CONSIDERATIONS

Scalp Injuries

The abundant blood supply and inability of the blood vessels to contract or constrict allows scalp lacerations to bleed more profusely than comparable wounds in other body areas. In infants and small children the loss of blood may produce shock and necessitate emergency replacement by transfusion. Scalp lacerations usually heal by first intention if properly cleansed, debrided and sutured.

Subgaleal hematomas are often perplexing, inasmuch as their soft centers and hard, raised edges may give the impression of an underlying depression of the skull. These hematomas must be distinguished from "spurious meningoceles," which are subgaleal collections of cerebrospinal fluid. The latter are due to an underlying fracture of the skull, accompanied by dural and arachnoidal tears that permit the extracranial escape of cerebrospinal fluid. Both of these collections usually disappear with the passage of time. At times, needle aspiration may be required.

Skull Fractures

In contrast to the skull of the older child and adult, which is relatively rigid and nonyielding, the skull of the infant is elastic and subject to considerable deformation. Its vault consists of rather loosely joined flat bones separated by sutures and fontanelles. As compared with the elastic vault, the base of the skull is relatively rigid. The posterior fontanelle is usually closed at the age of two months, the anterior fontanelle between the ninth and sixteenth month. The sagittal, coronal, lambdoidal and temporosquamosal sutures are not firmly united until the child is about four years of age.

Due to the elasticity of the vault of the infant's skull, depression of bone may occur without extensive fracturing, the fracture being of the so-called "ping-pong ball" or "derby-hat" type. Because of the loose joint at the suture lines, diastatic fractures are more common in children. The infant dura is rather rigidly attached to the inner table of the skull and is, therefore, commonly torn directly beneath a fracture line. As noted previously, this often permits the escape of cerebrospinal fluid beneath the scalp, forming the so-called spurious meningocele. Dural tears involving the major venous sinuses may produce subdural and extradural hematomas.

The more serious skull fractures are those in which there is comminution of bone. These comminuted fragments are frequently driven inward, producing a simple, depressed fracture or are associated with an overlying laceration of the scalp resulting in a compound, comminuted, depressed fracture. At times, the in-driven fragments will penetrate the dura and lacerate the underlying brain.

Presented before the Section on Pediatrics at the 89th Annual Session of the California Medical Association, Los Angeles, February 21 to 24, 1960.

In general, linear fractures in infants and children do not, per se, present a problem. Unless the edges are widely separated, most linear fractures heal quickly and are no longer apparent on radiological examination in three to six months. If the fracture line crosses an arterial or venous channel, the child should be closely observed for signs of increasing intracranial pressure that signal the formation of a subdural or extradural hematoma.

Linear fractures involving the base of the skull (basal fractures) may not be apparent on either clinical or radiological examination. The diagnosis is usually established by finding leakage of cerebrospinal fluid from the nose or ear. Fractures involving the paranasal sinuses may enable air to enter the intracranial chamber and produce a pneumocele or spontaneous ventriculogram. The presence of rhinorrhea, otorrhea or intracranial air converts a simple fracture into a compound fracture and requires appropriate antibacterial prophylactic therapy.

Brain Injury

Concussion, cerebral contusion and laceration, cerebral hemorrhage and edema, and meningeal hemorrhage in its various forms are common sequelae of cranial trauma in both children and adults. However, because more of the energy of the blow is absorbed by the deformation of the skull, the brain is relatively more protected from injury in infants than in adults. Some authorities point out that for a similar reason the brain stem of infants is less vulnerable.

The most common change in the human brain resulting from a head injury is the phenomenon known as concussion. Before World War II, concussion was considered to be a mysterious disturbance of cellular function which was unaccompanied by any demonstrable pathological change in nerve cells. However, the stimulus provided by the war led many investigators to reassess the brain injury problem, particularly the nature of concussion. It has now been shown that concussion produces swelling in nerve cells, chiefly those of the brain stem's reticular formation. Intensive studies of the function of the reticular formation have disclosed that this area has a modulating and integrating effect on motor and sensory function, autonomic balance, visceral function, alertnesss and sleep and the maintenance of consciousness. The diagnosis of concussion is established if there has been a disturbance of consciousness following a head injury. This may vary from momentary confusion to coma. Children with relatively minor head injuries may have what has been termed delayed concussion. This is characterized by drowsiness, pallor, vomiting and diaphoresis, coming on several hours after the injury. In the presence of these symptoms, close observation for signs of intracranial bleeding is required even though most often the child recovers promptly. How this delayed form comes about is obscure.

Frequently, the forces involved in cranial trauma will produce gross lesions in the central nervous system, such as contusion and laceration. In severe closed head injury, the frontal and temporal poles of the brain are subject to the greatest damage. This is due to shear strains that develop in these lobes because of the rotation of the brain within the skull at the time of impact. Cerebral contusion and laceration may occur directly beneath the site of the blow (*coup* injury) or at a point directly opposite to it (*contrecoup* injury).

Edema of the brain will develop in the wounded areas. Generalized cerebral contusion may result in such widespread edema that the intracranial pressure will reach critical or fatal levels. Reduction of this edema is imperative, inasmuch as the associated ischemia may produce widespread death of nerve cells.

Persisting neurological defects will occur as a result of irreparable brain damage. These paralytic phenomena will vary, depending on the location and extent of the injury. Convulsions will develop as a late manifestation in approximately 2 per cent of patients with closed head injury and in 20 per cent of the patients in whom the brain wound is associated with dural penetration.

TREATMENT

Emergency Measures

In the management of the child with a head injury, the four major steps to be taken are: Establishment of an adequate airway followed by the administration of oxygen; control of shock; control of hemorrhage; assessment of the extent of injury.

Establishment of an adequate airway is the first and most important step. Patients with head injury commonly have respiratory obstruction due to depression of the gag, cough and swallowing reflexes coincident with the accumulation of the pharyngeal secretions. During transport to a place of treatment the child should be placed in a modified prone position to promote gravity drainage. If suction is available, a soft rubber or plastic catheter is preferable to a metal device that might contuse the throat. If these measures do not establish a good airway, direct laryngoscopy, bronchoscopy or tracheotomy may be necessary. Neurosurgeons have used tracheotomy with increasing frequency in the past decade.

Shock is uncommon in uncomplicated head injuries and, when present, is usually the result of

intrathoracic or intra-abdominal injury or fractures of long bones. However, particularly in infants and small children, shock may occur as a result of loss of blood. It is good policy to start an intravenous infusion immediately in all seriously injured patients and arrange for blood typing and cross-matching on the assumption that blood replacement may be necessary.

Hemorrhage from scalp wounds may be profuse, particularly if the larger arteries have been lacerated. Bleeding from most scalp lacerations may be controlled by compression dressings owing to the fortuitous anatomical arrangement of a bony backstop afforded by the underlying skull. Hemostats can be applied to bleeding vessels and incorporated in the dressing. Scalp and other wounds should not be sutured until the condition of the child permits this measure.

Having established measures to control respiration, shock and hemorrhage, the physician can then make an assessment of the extent of injury. From the neurological standpoint, evaluation of the state of consciousness is of paramount importance. Disturbed consciousness may vary from confusion to coma. After investigation on this point, the examiner should proceed with a systematic neurological examination, the completeness of which will depend on the responsiveness of the patient. The size, equality or inequality and reaction of the pupils should be noted. Ophthalmoscopic examination is rarely helpful but may disclose acute papilledema or retinal hemorrhage. The ears and nose must be examined for evidence of leakage of cerebrospinal fluid. Seventh nerve involvement may be noted by close observation of facial grimaces. Since neck injury occurs in about 25 per cent of all patients with serious head injury, this area should be carefully examined. The extremities should be observed for signs of weakness or paralysis. Observing the movements of the restless patient often will help in this determination. In a semicomatose patient, movement may be induced by applying a painful stimulus to the hands and feet. The activity of the abdominal and deep tendon reflexes and the plantar responses should be noted. Evaluation of the various sensory functions will depend on the alertness and cooperation of the patient.

Assessment of the neurological status in the immediate post-traumatic period is of paramount importance, inasmuch as it enables the examiner to establish a baseline. A steady improvement in the state of consciousness and the disappearance of abnormal neurological signs is reassuring and usually indicates a good prognosis. Conversely, progressive loss of consciousness accompanied by pupillary inequality, hemiparesis, slowing of the pulse and respiration and an increase in pulse pressure implies that either intracranial hemorrhage or cerebral edema is developing.

Radiological examination is indicated in all patients with head injuries but is not justified as an emergency measure unless it can be done without jeopardizing the patient's chances for recovery. X-ray studies may disclose linear or diastatic fractures which may be of localizing value should an extradural or subdural hematoma develop. The pattern of simple and compound, comminuted, depressed fractures enables the surgeon to plan the operative procedure.

Lumbar puncture is not recommended as a routine diagnostic procedure in patients with head injuries. In performing this test, the attending physician wishes to determine if there is elevation of intracranial pressure and/or blood in the cerebrospinal fluid. Measurements of cerebrospinal fluid pressure are often inaccurate due to the restlessness and straining of the patient or respiratory obstruction or a block in the cerebrospinal fluid pathways. Cerebrospinal fluid blocks may occur at the tentorial notch and the foramen magnum due to shifting of intracranial structures brought about by the pressure of large subdural and extradural hematomas. If this state exists, lumbar puncture may permit further strangulation and hemorrhagic infarction of the brain stem.

In general, lumbar puncture is done to determine whether or not there is blood in the cerebrospinal fluid. Subarachnoid hemorrhage implies that cerebral contusion has occurred, which influences the prognosis and treatment. Patients with meningismus secondary to subarachnoid bleeding may be symptomatically improved by withdrawing 5 to 15 cc. of cerebrospinal fluid at intervals. However, repeated lumbar puncture does not result in a more rapid disappearance of the red blood cells.

TREATMENT

General Measures

The most important part of the program in the management of a patient with a head injury is close observation of the state of consciousness, the vital signs, the size of the pupils and movement of the extremities. The nurses in attendance should be instructed in making these observations. Oxygen and suction equipment should be available at the bedside. Routine and special orders by the physician are outlined in Table 1.

Conscious patients are given clear liquids in whatever amount they wish during the first 24 hours. At the end of this time, solid food and fluids are given as desired. In unconscious patients fluids are administered intravenously in amounts of approximately 10 cc. per pound of body weight per day.

As the state of consciousness improves gavage feedings may be started unless there is a persisting rhinorrhea, serious rhinopharyngeal injury or persistent vomiting associated with depressed pharyngeal reflexes.

Hypertonic intravenous infusions are used only if proven cerebral edema has produced a significant elevation of intracranial pressure. Hypertonic glucose and sucrose solutions are available in most hospitals. Concentrated plasma and serum albumin are effective agents but quite expensive. In recent years, intravenous administration of urea in a 10 per cent solution of invert sugar has been used with increasing frequency. It must be remembered that most of these dehydrating agents have only a temporary effect, which may be followed by a "rebound phenomenon" during which the intracranial pressure may exceed that which existed before they were administered.

Patients with head injuries are usually placed in a position of head elevation to promote venous drainage. However, some neurosurgeons prefer to keep their patients flat. Unconscious patients should have their position changed every two hours to discourage the development of skin and respiratory complications.

Drugs

Analgesics and sedatives that do not significantly impair the state of consciousness or depress respiration may be used. Aspirin may be used freely, and in general it will control headache and restlessness. For more severe pain, hypodermic injection of small amounts of codeine or meperidine (Demerol) may be required. Barbiturates are used to control restlessness. Paraldehyde administered intramuscularly or rectally is probably best for controlling extreme restlessness in children.

Antibacterial therapy is not instituted as a routine measure. It is indicated when there has been extensive wounding with contamination, in semicomatose and comatose patients with depressed pharyngeal reflexes and in patients with cerebrospinal fluid fistulas.

Anticonvulsant measures will be required to combat acute seizures. Barbiturates and hydantoin derivatives are used. It has been our policy to keep all patients with brain wounds on anticonvulsant medication for 12 to 18 months after the injury. If seizures do not develop, the dosage is gradually reduced over this period of time.

Control of Body Temperature

A significant rise of body temperature may occur in patients with cerebral contusion accompanied by hemorrhage and edema. In these damaged areas are nerve cells that will either live or die, depending on the amount of oxygen that is available to sustain their metabolic rate. Reduction of body temperature decreases the oxygen demand and the metabolic rate of these damaged cells, and reduces intracranial pressure by protecting against cerebral edema.

Body temperature of 102° to 103° F. can usually be reduced to normothermic levels by the use of salicylates, alcohol sponges, ice bags, ice-water enemas and the exposure of the patient undressed to air being circulated by a fan. In recent years, more vigorous hypothermic methods have been employed in the management of patients with severe head injury. These patients will often have rectal temperatures of 105° to 107° F., generalized rigidity and recurrent opisthotonic episodes accompanied by tachycardia and tachypnea. By the use of ice packs or special hypothermic apparatus the body temperature is reduced to a level of 90° to 95° F. In addition to these physical measures, the drugs of the "lytic cocktail" (chlorpromazine, promethazine, meperidine and levallorphan) (Table 2) are administered intravenously and intramuscularly to enhance the effect of the externally applied cold.

TABLE 1.—*Routine and Special Orders by Physician to Nurses Dealing with a Patient with Head Injury*

ROUTINE:

1. Blood pressure, pulse and respiration rate every 30 minutes until responding—then every two hours.
2. Rectal temperature every two hours.
3. Check ability to rouse, pupils and hand grips, every two hours.
4. Elevate head of bed 10 to 12 inches.
5. Put side rails on bed.
6. Administer oxygen constantly until responding. Keep airway clear with suction.
7. Nothing by mouth.
8. Sponge body with alcohol for temperature over 102° F.
9. Sedatives and analgesics.

SPECIAL:

1. Special nurses.
2. Hypothermia routine.
3. Tracheotomy routine.
4. Antibacterial therapy.
5. Intravenous isotonic and hypertonic fluids.
6. Blood transfusions.
7. Miscellaneous.

TABLE 2.—*Lytic Cocktail for Use in Treatment of Infants and Children with Head Injuries*

1. CHLORPROMAZINE HYDROCHLORIDE. (Thorazine)
   To reduce shivering.
2. PROMETHAZINE HYDROCHLORIDE. (Phenergan)
   Cutaneous vasodilator.
   Antihistaminic.
   Anticholinergic.
   Bronchial dilator.
3. MEPERIDINE HYDROCHLORIDE. (Demerol)
   Analgesic.
   Sedative.
   Spasmolytic.
   Cutaneous vasodilator.
4. LEVALLORPHAN TARTRATE. (Lorfan)
   Overcome drug-induced respiratory depression.

Evidence is now accumulating to indicate that these vigorous hypothermic methods are not only saving lives but restoring patients to useful positions in our society.

Electrolytes

Estimation of the serum electrolytes should be done frequently in all cases of severe head injury. Potassium levels tend to fall steadily in the presence of extensive cerebral injury. Hemoglobin values should be determined every four days, inasmuch as comatose and semicomatose persons tend to develop progressive anemia.

Activity

Ambulation is encouraged as soon as the condition of the child permits. If signs of fatigue become manifest, appropriate rest periods are instituted.

SKULL FRACTURE

Linear Fracture

Simple linear fractures of the vault require no surgical treatment. Basal fractures with rhinorrhea or otorrhea require prophylactic antibacterial therapy which is continued for at least three days after the cerebrospinal fluid fistula closes. Otorrhea rarely persists beyond the acute phase of the head injury, but rhinorrhea may become chronic and necessitate surgical intervention consisting of a reinforced closure of the dural laceration and repair of the defect in the floor of the skull with bone or one of the acceptable surgical metals or plastics.

Depressed Fracture

Small, shallow, depressed skull fractures may require no surgical treatment and will disappear with the growth of the skull. Larger depressed fractures should be elevated as soon as the condition of the child permits. "Ping-pong" fractures are easily managed by placing a marginal burr hole and pushing the bone outward with a blunt periosteal elevator. Comminuted, depressed fractures are managed by removing all the in-driven fragments.

Formerly, it was considered unwise to carry out a primary repair of the skull defect remaining after debridement of a compound depressed skull fracture. However, since the advent of antibacterial therapy replacement of the larger bone fragments has been carried out with excellent results from both the cosmetic and structural standpoints. If the wound is potentially infected, all of the bone fragments should be removed and the skull defect repaired later, using one of the accepted techniques for cranioplasty.

Brain Wounds

Brain wounds are treated by removing all devitalized tissue with suction. All in-driven bone fragments and foreign bodies are removed. Care is taken not to injure nervous tissue that is either functioning or may regain function. The dura overlying a brain wound is always closed in a water-tight manner. At times, this necessitates the use of a graft of temporalis fascia or periosteum.

Extradural and Acute Subdural Hematoma

Extradural and acute subdural hemorrhages constitute real surgical emergencies. The severe brain compression produced by these lesions may develop so rapidly that the patient dies before surgical measures can be begun. It must be remembered that in infants and young children extradural hemorrhage commonly occurs in the absence of skull fracture. The classical lucid interval may not occur. Furthermore, extradural hemorrhage may occur in the anterior and posterior fossae of the skull due to laceration of meningeal arteries or tearing of the venous sinuses.

Extradural and acute subdural hematomas are characterized by a more or less rapid deterioration of the state of consciousness, followed by pupillary inequality with dilatation of the ipsilateral pupil, slowing of pulse and respiration, increasing pulse pressure, rise of body temperature, progressive hemiparesis and, at times, generalized convulsions.

Acute Cerebral Edema

Generalized cerebral contusion with edema may produce a syndrome which is indistinguishable from that resulting from acute intracranial hemorrhage. Intracranial pressure may rise to critical levels. In such cases, standard trephine exploration in the frontal, temporal, parietal and suboccipital regions will disclose scattered areas of cerebral contusion without clot formation. Extensive bilateral subtemporal decompressions will often prove to be life-saving measures.

Chronic Subdural Hematoma

Chronic subdural hematoma is a comparatively late manifestation of head injury in infants and children. Intracranial hemorrhage of this type is due either to tearing of veins that extend from the surface of the brain to the major venous sinuses or to direct laceration of the venous sinuses. Venous injuries of this kind may occur in passage of the head through the birth canal, from maneuvers carried out to aid delivery or from a blow on the head.

In the acute phase the clot that forms is rather firm and bright red. With the passing of time it breaks down to form a more liquid, chocolate-brown mass. In the later phases it consists of clear xanthochromic fluid. Chronic subdural hematomas become surrounded by a membrane of varying thickness. These older hematomas will enlarge stead-

ily due to the osmotic attraction of the clot for cerebrospinal fluid.

The signs and symptoms of chronic subdural hematoma have no characteristic pattern. The lesion should be suspected in infants who have a bulging fontanelle, progressive head enlargement, listlessness, instability, poor appetite, vomiting, convulsions, hemiparesis and hyperactive deep tendon reflexes in varying combination. In older children, separation of the sutures, rather than a bulging fontanelle, may be the first sign.

Because these clots interfere with the growth of the brain, they should be recognized and treated as soon as possible. In the early stages, evacuation of the hematoma is accomplished by repeated needle aspiration of the subdural space through the lateral angle of the fontanelle. In the later stages, trephine exploration and drainage of the subdural space is the procedure of choice. If a well-developed membrane has formed, craniotomy is done and all solid clot and the membrane are excised.

744 Fairmount Avenue, Pasadena (Pudenz).